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Mr. Chad Rook  
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Mr. Rook:

I have reviewed the records you have forwarded to me. In accordance with your request, I am providing my opinions concerning what role, if any, asphyxiation may have played in the death of Mr. Teddy Parker.

The materials I have reviewed thus far are:

1. Videos of his arrest and events in jail
2. Two videos prepared by plaintiff's expert
3. EMS/ER/Hospital records
4. Autopsy Report
5. Preliminary Death Certificate
6. Written statements of eyewitnesses
7. Depositions of the Smith County employees
8. Reports of experts (Dr. Freeman and Dr. Valentine)
9. Plaintiff's original complaint
10. Excel Spreadsheet of Mr. Parker's ventilation
11. Visual screengrab of waveform

As background information, I am one of only a handful of physicians and the only physician specifically trained in pulmonary medicine and exercise physiology in the United States who has, through direct experimentation, investigated the physiologic effects of restraint and their effects upon ventilation, cardiac output, carbon dioxide (CO<sub>2</sub>) levels, and blood oxygen levels. The information I am providing you is based upon my training in this field, my own research, and the publications of other individuals who have conducted experiments utilizing recognized procedures and objective end

**EXHIBIT "H"**

points. I have also attached a list of cases in which I have provided testimony in the last 4 years as well as my fee schedule.

I am a physician licensed to practice medicine in the State of California. I was a full time member of the faculty of the University of California, San Diego until July 1, 2006. At that time, I was a Professor of Medicine and Surgery at UCSD and the Associate Director of the Department of Emergency Medicine. As of that date, I partially retired and now hold the position of Emeritus Professor of Medicine. I am still active in ongoing research in the field of restraint associated deaths. I am Board Certified in Internal Medicine, Pulmonary Disease, Occupational Medicine, and Undersea and Hyperbaric Medicine. Until January 1, 2017, I was also board certified in Emergency Medicine, however at that time I allowed my Emergency Medicine certification to lapse. These are all specialties recognized by the American Board of Medical Specialties. I do not feel it is necessary for me to further describe my activities and achievements as they are fully described in my curriculum vitae.

Based upon the material I reviewed, more important facts appear to be:

1. Mr. Parker was arrested due to what appeared to be bizarre behavior
2. During transport to jail his behavior alternated between compliance and agitation with probable delusional thought
3. When brought to jail the staff attempted to remove his earrings
4. His earrings could not be removed in the first area in which he was placed.
5. He was brought to a padded cell, seated, and further attempts to remove his earrings were made by the staff.
6. During these attempts, his behavior again alternated between compliance and agitation with resistance
7. During the attempts to remove the earrings, Mr. Parker spat on one of the officers. (Approx 10::39 on the video)
8. During the approximate minute prior to spitting on one of the officers, Mr. Parker's respiratory rate was about 44 bpm
9. As a result of his spitting, Mr. Parker's shirt was pulled up over his face (Approx 10::50 on the video).
10. From about 10::50 to 11::45 Mr. Parker's respiratory rate was approximately 47 bpm
11. From about 11::50 to 12::45, Mr. Parker's respiratory rate was approximately 33 bpm

12. There is variability in the duration of these breaths throughout this period compatible with his vocalizations and struggling
13. At no point during this period of time could Mr. Parker be heard to say he couldn't breathe, he was short of breath, nor any similar complaint.
14. At approximately 12::50 on the video Mr. Parker's last breath can be heard.
15. Fifteen to twenty seconds later he is noted to be no longer breathing and noted to be unconscious.
16. At this time he is also noted to be cyanotic
17. Resuscitation was begun and a ROSC noted
18. Paramedics arrived and continued life support.
19. He was brought to the hospital, where among other abnormalities he was noted to have a profound metabolic acidosis, a creatinine of 2.1, elevations of CK, (and CK-MB and troponin), evidence of a consumptive coagulopathy, a temperature of 103.7° F, and hyperkalemia,
20. On urine tox screen the presence of methamphetamine, amphetamine, alcohol, and cocaine were all noted.
21. In spite of maximal care, support was withdrawn approximately 3 days later.
22. An autopsy was performed which revealed a slightly elevated heart weight (360 grams), 50 to 80% stenosis of the proximal left anterior descending artery, poor dentition, and gingival erosions
23. Also noted at post mortem was a level of 1.85 mg/L of methamphetamine in Mr. Parker's blood.
24. The Medical Examiner's conclusion was Mr. Parker died of the toxic effects of methamphetamine.

Before addressing the question you asked of me, one must understand in circumstances such as these, asphyxiation, should it occur, would be due to inability to ventilate (breathe) to such a degree that blood oxygen levels fall, which ultimately causes the heart to stop. This is a process that in general, takes at least five minutes of no breathing at all (using an aspiration model such as drowning) because this process requires time for the oxygen stores in the lung to be depleted and then for the oxygen level in the blood to fall so low as to cause the heart to stop. Pure asphyxiation (without aspiration; as in a hanging or a therapeutic misadventure) takes considerably longer. Minor alterations in the ability to breathe (such as someone with mild asthma) or minor falls in blood oxygen levels (if you go to Aspen to go skiing your

blood oxygen level will fall by approximately 8%) are not clinically relevant.

The specific issue you asked me to address has several components to it. Firstly one must address the question of whether asphyxia could reasonably occur based upon the sequence of events in this case and does the basic physiology of ventilation predict any hypoxemia (low blood oxygen) would occur in this circumstance. Finally given the circumstances, what is the most likely clinical cause of death.

Given what appears to be the sequence of events in this case, asphyxiation is highly improbable. Mr Parker was alternating between cooperative behavior and struggling with agitation during the critical time period of this incident. For an individual to suffer a cardiorespiratory arrest from asphyxiation would (as noted above) require at least 5 minutes of *no breathing at all* for which there is no evidence in this case. Mr. Parker was breathing throughout the period in the padded cell until the time he suddenly stopped breathing. At no point did he complain he was having trouble breathing nor that he was short of breath and clearly he would have been able to speak given the ventilatory sounds audible on the video of the event. During the minute before his cardiorespiratory arrest Mr. Parker's respiratory rate was approximately 33 bpm. This respiratory rate is perfectly compatible with providing normal blood oxygen and CO<sub>2</sub> levels and compatible with his degree of struggling during this period of time. A normal respiratory rate at rest is about 14-16 bpm and his respiratory rate during that minute was double this rate. There is no evidence Mr. Parker could not breathe at all during the 2 minute period his shirt was lifted over his face (he could be heard to be breathing throughout this period and as noted above at no point did he complain he was short of breath or he could not breathe properly) but even if this were the case, two minutes of no ventilation is insufficient to cause a cardiorespiratory arrest or even any clinically important reduction in blood oxygen levels.

At the time he was noted to have stopped breathing he also appeared to be cyanotic. Cyanosis is a bluish discoloration of the skin. When it is due to asphyxiation, cyanosis would be expected to occur a noticeable period of time before a cardiorespiratory arrest occurred. It also appeared Mr. Parker was conscious essentially right up to the time he stopped breathing. This is not consistent with a case of simple asphyxiation. As the blood oxygen level falls from asphyxiation an early manifestation of low blood oxygen would

be cyanosis and at about that time cerebral functioning would diminish (prior to the loss of consciousness). Only after that would unconsciousness ensue. However, even after losing consciousness breathing would continue for a period of time. Mr. Parker lost consciousness, became cyanotic and had a cardiorespiratory arrest all within several seconds. This is not compatible with asphyxiation but is compatible with a sudden cardiac arrhythmia.

The notion minimal loads upon the chest or minimal upper airway obstruction will cause hypoxemia (low blood oxygen) in circumstances such as these, by sufficiently limiting ventilation, is based upon basic misunderstandings of ventilatory physiology. I cannot emphasize strongly enough that without the ability to quantitatively assess the effects of ventilatory loading and the oxygen requirements in any given situation, any expert's comments about asphyxia, difficulty breathing, or restraint of breathing must be viewed as *ipse dixit*. One can easily estimate the oxygen requirements for a person in Mr. Parker's circumstances based upon published data to assess his ventilatory needs in this setting. Clearly his ventilatory rate was more than sufficient to address these needs.

From a clinical point of view, in the setting of asphyxiation, not only does blood oxygen fall, but the blood CO<sub>2</sub> level rises dramatically. The first measurement of blood CO<sub>2</sub> taken at the hospital does not suggest asphyxiation occurred. Indeed, this blood test alone strongly argues against asphyxiation. An additional clinical point to consider is whether Mr. Parker was suffering from the excited delirium syndrome (EDS) at the time of his interaction with the police. Mr. Parker's alternating behavior, diaphoresis, elevated temperature, and multiple laboratory abnormalities noted early in his hospitalization are all more consistent with EDS than with simply being "high" from his use of methamphetamine; regardless of the blood levels of methamphetamine. The autopsy suggests Mr. Parker was a long term abuser of methamphetamine and EDS is most frequently seen in chronic abusers of stimulant drugs and often with relatively low blood levels of the active drug. Regardless, there is no "safe" level of methamphetamine. In general, in the Emergency Department blood levels of such drugs are not measured, as the important issue is the toxic manifestations of the drugs and not a "blood level." Mr. Parker's behavior and physiologic abnormalities all point to the severity of his intoxication. Additionally cocaine and alcohol undoubtedly contributed to this toxidrome. However this doesn't alter the basic physiology of Mr. Parker's death which

was ultimately related to the toxic effects of methamphetamine and the other drugs he was using.

Upon admission to the Emergency Department, Mr. Parker had a number of laboratory abnormalities which indicated severe drug intoxication. He had a profound metabolic acidosis, hyperkalemia, evidence of rhabdomyolysis, and evidence of acute renal failure. None of these abnormalities can be ascribed to either a recent simple asphyxiation or his relatively brief cardiac arrest, but are all indicative of a severe drug intoxication. He also had evidence of a consumptive coagulopathy which may have been the cause of the muscle hemorrhage noted at autopsy, as there was no evidence of diffuse muscle trauma on the videos I reviewed, nor are there any statements to the effect physical strikes were necessary to control Mr. Parker's behavior.

One must also consider the severe coronary artery disease found at Mr. Parker's autopsy. An 80% stenotic lesion of the left anterior descending artery is referred to as the "widow maker." I think the meaning of this term is self explanatory. Although atherosomatous abnormalities are indeed common in men of Mr. Parker's age, a 80% LAD lesion is distinctly unusual and is definitively associated with a high risk of sudden death. Coronary lesions of this sort are more commonly seen in chronic abusers of stimulant drugs. Individuals with this sort of lesion who are undergoing physical exertion have a 150 fold increase in the risk of sudden death. Sadly, these abnormalities were not identified at Mr. Parker's cardiac catheterization, but I doubt even had they been recognized, that the outcome would have been different.

Thus the most likely explanation in this case is the mechanism of death was that of a lethal cardiac arrhythmia in the setting of acute EDS caused by methamphetamine (and other drugs) and underlying medical problems most importantly his CAD and his profound metabolic acidosis.

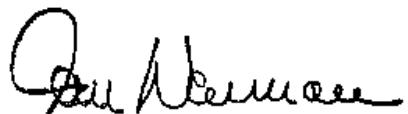
In summary then, the clinical diagnosis in this case is not that of an asphyxial death. The description of the event as well as the arterial blood gas present a picture incompatible with asphyxiation and our day to day experience with masks covering the mouth and nose also suggests this was not a case of asphyxiation.

Thus my primary opinions (to a reasonable level of medical certainty) are:

1. Mr. Parker did not die of asphyxia.
2. Asphyxia did not contribute to Mr. Parker's death.
3. Mr. Parker's death was due to a lethal arrhythmia in the setting of an agitated state (likely the EDS) induced by his drug abuse and complicated by his CAD and other medical problems.

These opinions are based upon the information so far supplied to me and further accounts of the events that took place and additional medical records may alter my final opinions. Should addditional material become available, I must reserve the right to amend my opinions. I look forward to reviewing any additional testimony or material, which might affect my opinions.

Very truly yours,



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